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The Biology of the Eosinophil Leucocyte

BY

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SUMMARY.—A brief review is given of the structure, distribution, fate, and functions, of eosinophil leucocytes. A hypothesis attempting to explain the occurrence of eosinophilia or eosinopenia of circulating blood is presented. It is suggested that the disposition of eosinophils within the body is dependent upon local histamine concentration since eosinophils are chemotactically responsive to histamine.

Description

IN 1846 Wharton Jones gave the first recognisable description of an eosinophil leucocyte. He used a Ross compound microscope with an $\frac{1}{8}$-inch objective and his examination included, among others, the blood of the horse (Fig. 1). He says that he studied the blood "no more than 2 days old," and he did not mention the use of any anticoagulant. In spite of these almost incredible disadvantages, he gave a very fine description of what he called the "coarse granular cell" of blood, which is without question a description of what we know as an eosinophil leucocyte. In 1879 Ehrlich described "acidophil leucocytes" as a result of the discovery that these particular leucocytes had great avidity for acidic dyes. It was not until 1925 that Cunningham, Sabin, and Doan, working with rabbits, described the origin of eosinophil leucocytes in the bone marrow. They gave beautiful descriptions and drawings of a complete developmental series, and this concept of the development of eosinophils in bone marrow has never since been seriously challenged.

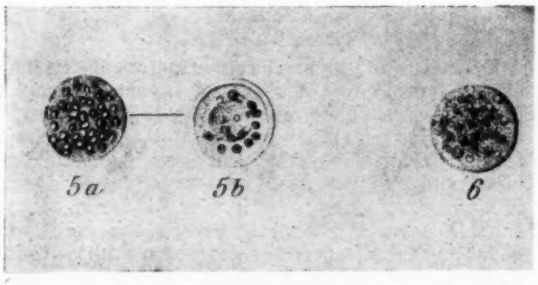

FIG. 1.—Original drawing by Wharton Jones (1846) of "Coarse granular leucocytes." (*Phil. Trans. R. Soc. Lond.* **1.** 63.)

As it occurs in circulating blood, the eosinophil is normally an adult or fully mature cell. It usually has a bilobed nucleus and the whole of the cytoplasm is filled with a large number of spherical granules (Figs. 4 and 7). These granules stain yellow with eosin and with most of the so-called panoptic stains in use, such as Leishman's, Wright's, or Giemsa's stain. The adult cells arise from an undifferentiated myeloblast of the bone marrow, which becomes differentiated into the eosinophilic series at the promyelocyte stage. At first it is possible to make out from two to twelve very small eosinophilic granules, but later 20 or 30 fine granules are visible (Figs. 2 and 5). As the leucocyte matures through its series (myelocyte, metamyelocyte, and staff forms), these granules increase in size and number (Figs. 3 and 6). The maximum number of granules is seen in the myelocyte, in each of which there are between 100 and 170 granules per cell *in equidae*. In the adult there are no more than about 35 and this loss is attributable to mitotic division which occurs mainly at the myelocyte stage. Cytochemically, eosinophil leucocytes differ from other leucocytes of blood in the structure and composition of the granules. These spherical granules are coated by a phospholipid compound which will readily stain with sudan black and other fat-soluble stains. The centre contains a protein which is rich in arginine, the enzyme peroxidase and a compound which exerts an antihistaminic effect. The latter may well be the same as the arginine-rich protein (Vercauteren, 1953). Study of eosinophils using the electronmicroscope (Goodman *et al.*, 1957) showed that eosinophil granules contain crystals. These crystals, however, have not been seen except by electronmicroscopy and it is not possible at the present time to be certain whether or not they are artefactual. It is possible that

some of the contents of the eosinophil granules are caused to crystalise by the fixation method used in the preparation of specimens for electronmicroscopy. There is, however, some evidence that there are two distinct kinds of eosinophil granules. When these cells are stained by Bakers acid haematin method (Baker, 1946), some of the granules, particularly in the more primitive eosinophils, fail to take up the stain. On some smears stained with Leishman stain some of the granules in the primitive cells are basophilic rather than eosinophilic and under the electronmicroscope some granules fail to show crystal formation. If indeed two kinds of granules really exist in these cells, then it is at least certain that the more common kind is heavily predominant and there is no satisfactory evidence of variation in the granules of the normal adult cell.

Control of Production and Release

Eosinophilia

Increases in the number of eosinophils of the blood and tissues have for a very long time been associated with parasitic infestation. In 1904 Opie described in man the lesions caused by *Trichinella spiralis* in muscle. These lesions were surrounded by eosinophil leucocytes in enormous numbers. Craig and Faust (1943) described many infestations, and in some of these eosinophil counts as high as 10,000 per c.mm. have been recorded in the blood of man. There is no demonstrable direct correlation between the parasitic burden of the bowel and the circulating eosinophils. There is, however, strong evidence that infestation does increase the eosinophilic content of the body as a whole, the greater part of the cells being found in the bone marrow and in the tissues which are invaded by the parasites in question. Eosinophilia of blood as a result of parasitic infestation is not a constant finding, since the cells which are circulating are very readily subject to removal from the blood as a result of stress or by reason of the fact that they spend only a short part of their life in the circulating blood. Eosinophilia also occurs in allergy and as a result of antigen-antibody reactions in tissue. In these cases the bone marrow eosinophils are usually increased, and also those in the local tissue involved, but the blood may be eosinopenic because of the withdrawal of the cells from the blood to the tissues affected. If, however, the allergic condition is prolonged more than a few hours, blood eosinophilia will occur.

Eosinophilia of tissue may occur in the more chronic forms of inflammation. Eosinophils are not typical of acute bacterial inflammations, nor are they common in pus. The cells are essentially a characteristic of lower grades of inflammation as, for example, may be caused by migrating parasitic larvae. In such lesions as the latter, eosinophil leucocytes frequently form a cuff outside an area infiltrated with small round cells around the path of the larva.

Eosinopenia

Eosinopenia of circulating blood is a feature of the alarm reaction described by Selye (1949). It also occurs in shock, and when there is any increase in the corticosteroid output of the suprarenal gland or, of course, an increase of steroids attributable to injections of these compounds (Hills, Forsham & Finch, 1948; Thorn *et al.*, 1948; Cape, 1952).

Intravenous injections of histamine are followed by an eosinopenia of circulating blood (Halpern & Benos, 1951; Panzenhagen & Speirs, 1953; Archer, 1956). This apparently paradoxical fact is probably capable of explanation as a result of the observation (Rose & Brown, 1938; Rose, 1940) that intravenous injections of histamine are followed within minutes by a decrease in the normal blood histamine levels and that a rise is impossible to achieve for more than a few moments as a result of a single intravenous injection.

Godlowski (1951) suggested that the corticosteroid hormones of the suprarenal gland were directly lytic to eosinophil leucocytes. As a result of work on dogs, he also suggested that eosinophil leucocytes were developed in the bowel wall. This, however, has not been accepted. Romani (1952) postulated a tissue migration of eosinophils in the local alarm reaction. He suggested that the eosinopenia which accompanied this reaction was caused both by the removal of eosinophils from blood to the affected tissue and by a lytic action of the hormones concerned on these cells. However, in 1954, Esselier *et al.* postulated that there was an increased reticuloendothelial destruction of eosinophil leucocytes together with an inhibition of the bone marrow, so that an eosinopenia of blood was produced. Dustin and de Harven (1954) postulated that corticosteroids might act as a mitotic poison to eosinophils and also that they have a short life in circulating blood. Panzenhagen and Speirs (1953), working with mice to which they had injected horse serum, showed that when they injected cortisone intraperitoneally into these animals there was firstly an eosinopenia of the blood and then, some hours later, eosinopenia of the peritoneal fluid. Quittner *et al.* (1951) and Esselier and Wagner (1952) showed that there was no change in the bone marrow content of eosinophils when there was a circulating eosinopenia attributable either to ACTH or to cortisone. This finding was confirmed and it was shown that, when the eosinopenia-producing agent was withdrawn, the bone marrow eosinophils increased in number before the blood eosinophils (Gross, 1957; Archer, 1957).

Duration of Life

By means of tissue culture experiments Osgood (1937) showed that the life of human eosinophil leucocytes was of the order of 8 to 12 days. Dustin and de Harven (1954) postulated that only about four hours of that period was spent in circulating blood and this hypothesis has received a good deal of indirect experimental support. There is only a small degree of diurnal change in the eosinophil count and there is no other short-term cyclical variation in the blood content of these cells in the normal animal. The degree of eosinopenia produced by therapeutic doses of corticosteroids is of the order of 60 per cent. within four hours. The evidence at present agrees with Osgood's estimate of the total

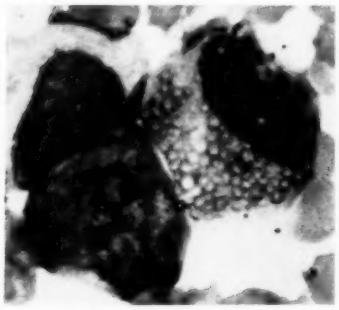

FIG. 2.—Late eosinophil promyelocyte. Leishman stain × 1,200.

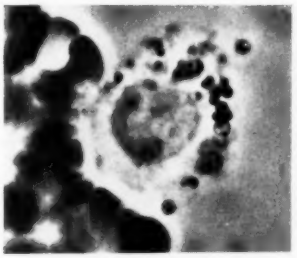

FIG. 5.—Intermediate eosinophil promyelocyte. Living cell, phase contrast × 1,200.

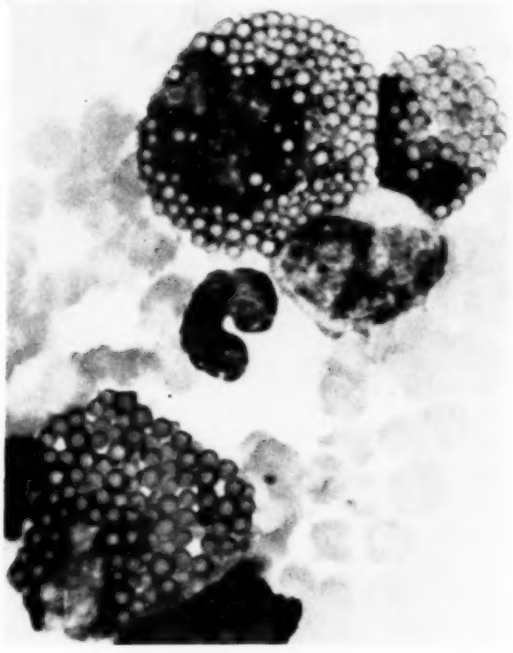

FIG. 3.—(From above.) Eosinophil myelocyte and metamyelocyte. Leishman stain × 1,200.

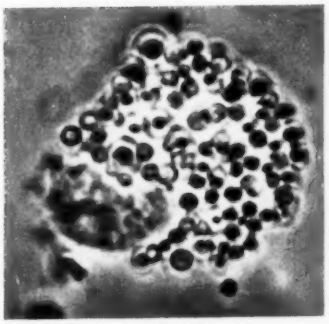

FIG. 6.—Eosinophil metamyelocyte. Living cell, phase contrast × 1,200.

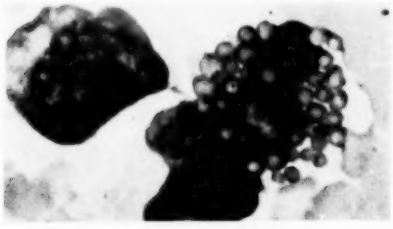

FIG. 4.—Eosinophil adult (bilobed polymorph). Leishman stain × 1,200.

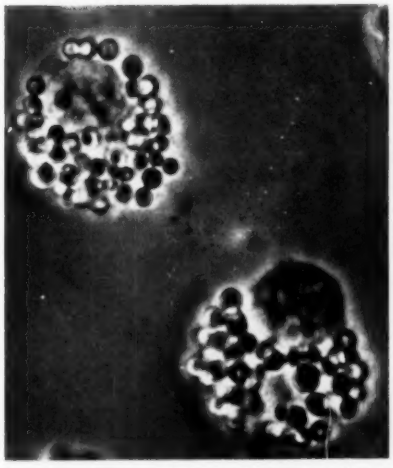

FIG. 7.—Two adult eosinophil bilobed polymorphs. Living cell, phase contrast × 1,200. (One lobe of the nucleus in each case is out of the plane of focus.)

I. ZLOTNIK—A CASE OF FOCAL SYMMETRICAL ENCEPHALOPATHY IN A SHEEP

FIG. 1.—Mesencephalon, two symmetrical shadow plaques. Spielmeyer × 4.

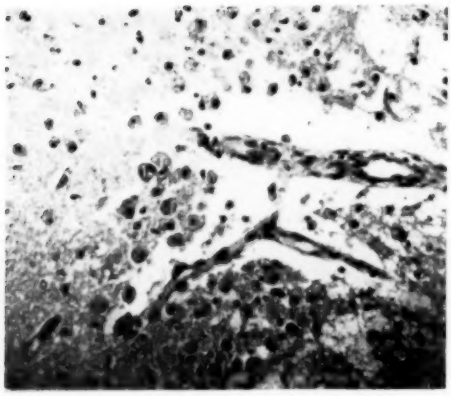

FIG. 2.—Proliferation of brain macrophages at the periphery of a shadow plaque. H. & E. × 150.

life of the eosinophil except that only a few hours of this are spent in circulating blood. In horse tissue it has been shown that eosinophils attracted to local areas of skin by histamine injections persist there for only three or four days (Archer, 1959).

Fate

There has been considerable speculation and no definite evidence as to the fate of eosinophil leucocytes. Lysis is unlikely, unless it occurs in stressed tissue. It does not occur as the result of an action directly or indirectly of ACTH or of the glucocorticoids produced by the suprarenal gland. Lysis is not produced by histamine. When eosinophils become senile they may lose their granules, so that they might become remarkably like lymphocytes. It is possible that they are destroyed by phagocytosis in the spleen or lymphatic glands, and some evidence to support this hypothesis has been produced by Speirs and his co-workers in New York (Speirs, 1958b).

Function

Eosinophil leucocytes respond chemotactically to histamine (Kline *et al.*, 1932; Knott & Pearson, 1934; Rieck, 1954; Archer, 1956), and an antihistamine effect of these cells has been demonstrated by *in vitro* experiments (Kovacs, 1950; Vercauteren, 1953). Some evidence of an antihistaminic effect of eosinophils *in vivo* has been published (Archer, 1959). In tissue rich in mast cells and therefore rich in histamine a fall of histamine content occurs when an influx of the eosinophils takes place (Riley, 1956, 1959).

A limitation of the effect of histamine is an attractive hypothesis for the function of eosinophil leucocytes, since a local increase in histamine causes a local eosinophilia, and there is some evidence that as a result of this influx of eosinophils the concentration of histamine is reduced. It may be that a rapidly mobilised, first defence system is provided by eosinophils, whereby there is some limitation of the effects of local histamine release.

It would appear likely that eosinophil leucocytes have some further function or functions. Godlowski (1951) suggested that they might act as an enzymatic template for various purposes in the tissue. Speirs (1958a) has suggested that eosinophils are involved in antibody formation, since they are attracted to the antigen-antibody reaction site. There is also some evidence that 5-hydroxytryptamine can be antagonised by these cells (Cohen & Mines, 1959; Archer, 1959) and that they can exert some antihyaluronidase effect (Vercauteren, 1953). It can, however, be said that an antihistaminic effect of the cells is established, but that it is not yet established whether or not this is their main physiological function. Certainly eosinophil leucocytes are not themselves rich in histamine as was thought to be the case (Code, 1952). The chief source of body histamine would appear to be the mast cells of the tissue (Riley, 1959) and probably the somewhat similar basophils of blood. Since mast cells are very easily disrupted by local tissue damage and this will be accompanied by a local increase in available tissue histamine, the presence of eosinophils can be explained. Histamine has many physiological functions, and one of these is to attract eosinophil leucocytes to the site of histamine release.

A Working Hypothesis

Bearing these facts in mind it is possible at the present time to formulate a working hypothesis to explain both the occurrence of eosinopenia and eosinophilia of blood as a consequence of changes in local and circulating histamine concentration (Archer, 1958).

Local stress of relatively temporary duration, produced in tissue as a result of damage or of the antigen-antibody reaction, leads *inter alia* to a release of histamine. It follows that eosinophils will be attracted to the site and thus some of these cells will be removed from circulation. If the stress is sufficient there will be a local excess of histamine, some of which may be carried away in the blood stream. This may cause stimulation of the anterior pituitary to produce ACTH (Mialhe-Voloss, 1955), which, by stimulation of the suprarenal glands, causes an increase in circulating corticosteroids and this, in turn, produces an inhibition of the biogenesis of histamine (Schayer, Smiley & Davis, 1954; Halpern, 1956; Halpern & Briot, 1956; Hicks & West, 1958). There is also the effect of deaminating enzymes to be considered because it is known that relatively large amounts of histamine can be detoxified in circulating blood (Rose & Browne, 1938; Kahlson, 1956). From this it follows that the circulating histamine level will very soon be below normal and so the stimulus to release eosinophils from the bone marrow and, indeed, the stimulus for eosinopoiesis will be inhibited, so that there is a fall in circulating eosinophils.

If the local stress is considerable and prolonged, for example in parasitic invasion or in allergic states, then the circulating blood histamine may remain above normal and so cause increased bone marrow production and release of eosinophils. A peripheral eosinophilia would follow.

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(*Continued at foot of page* 158)

A Case of Focal Symmetrical Encephalopathy in a Sheep

BY

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SUMMARY.—A case in an adult sheep has been described in which symmetrically distributed partial degeneration of myelin was present in the mesencephalon and cortex.

THE purpose of this communication is to describe a degenerative condition of the brain hitherto unrecognised in the adult sheep. Although the clinical signs shown by the sheep suggested cerebral trauma, the pathological findings were similar to those of focal symmetrical encephalomalacia in the lamb.

Clinical Signs

A 16-month-old Cheviot hogg belonging to the Institute, which appeared normal when seen the day previously, was found recumbent one morning with a very large soft swelling on the head extending from the ears to both jaws. Abrasions were present on the lips and over the left temporal region. When raised the animal tended to press the head against the wall and when forced to walk showed definite inco-ordination of the forelegs. After 24 hours the swelling on the head began to subside but no improvement was noticed in the general condition. Two days later the sheep was prostrate, unable to rise, and it was destroyed the following day *in extremis*.

Pathology

Macroscopical Examination

Small cuts and excoriations were found on the lips and on the skin of the left temporal region. Small haemorrhages were present in the subcutaneous tissue of the forehead as well as in the brain meninges. Cross sections through the brain revealed small widespread haemorrhages in the cerebral cortex, mesencephalon, and pons.

Microscopic Examination

Marked lesions were found both in the mesencephalon and in the cerebral cortex. In the mesencephalon 2 elliptical plaques of equal size were situated on both sides about 2 mm. below the dorsal surface of the anterior colliculi of the corpora quadrigemina. The plaques measured approximately 5 × 2½ mm. each and were sharply demarcated in sections stained for myelin (Fig. 1). Within the plaques myelin was greatly reduced (shadow plaques) and the majority of neurones were either necrotic or degenerated and partly replaced by microglial cells. The periphery of the plaques was surrounded by a layer of cerebral macrophages containing both fat and unchanged myelin (Fig. 2). The neural tissue around the plaques was oedematous, and the astrocytes, especially those situated near capillaries, were greatly swollen. The majority of the capillaries showed evidence of proliferation and hyperplasia of endothelial cells. Haemorrhages were present throughout the mid-brain and these were either small perivascular or large extravasations of blood in the nervous tissue. (See plate facing page 157.)

In the cerebrum changes were confined to the gyrus mediales. The lesion was not so sharply outlined as that of the mid-brain, but a gradual loss of myelin was seen at the periphery of the central white matter with diffuse proliferation of capillaries and migration of fat-laden macrophages. In the adjoining grey matter several neurones were degenerated while the number of microglial cells was greatly increased.

Discussion

Although the swelling and the abrasions on the head could point to a traumatic injury the sym-

(*Continued at foot of col.* 1, *page* 159)

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Normal Serum Albumen Values in Nigerian Zebu Cattle

BY

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SUMMARY.—The seasonal and age variations of serum albumen values in White Fulani Zebu cattle are described. During the rains, the mean value for all animals studied was 2·48 g. per 100 ml. During the dry season considerable variation occurred and in April, at the end of the dry season, the mean value was 1·83 g. per 100 ml. Differences between age group were not significant while wet to dry season differences were. There was a tendency for a drop to occur from 6 to 12 months of age and for pregnant cows to have higher serum albumen levels than non-pregnant cows.

Introduction

PREVIOUS work has shown the usefulness of serum albumen values as a guide to condition and as a criterion of pathogenic effect in helminth infestations in the bovine animal : Ross, Lee and Armour (1959), Ross, Armour and Lee (1959), and Ross and Armour (1959). It was shown that a serum albumen level of 1·9 g. per 100 ml. or less, in calves of 6 to 12 months old experiencing their first helminth challenge during the rains, was generally associated with a pathogenic effect in helminthiasis. The present study was undertaken to obtain normal serum albumen levels for use in the assessment of pathogenic effect in helminth infestations.

Materials and Methods

Fifty-four White Fulani Zebu cattle, from the Vom Livestock Investigation Centre herd, were divided into 9 age groups of 6 animals at each observation. The 54 cattle were selected at random at each seasonal observation. The 9 age groups were as follows : 0 to 2 months, 6, 12, 18, 24, 30 and 36 months, and 2 groups of cows over 6 years old, one group being pregnant and the other non-pregnant. The selected animals were bled from the jugular vein in July, 1958, October, 1958, January, 1959, and April, 1959, the sera separated and stored for analysis at –4°C. The " non pregnant " cow group consisted of cows which had calved within the previous 6 weeks, while the " pregnant " cows group contained cows within 4 months of calving. In July, analysis of the 54 individual sera was carried out but on the other 3 collection dates (October, January and April) individual serum analysis was made on the 6 and 12 months old groups only. On the last 3 collection dates individual serum samples from the remaining 7 groups were bulked by mixing 1 ml. from each individual serum from the group. Total protein estimates were made on the individual serum and on the bulk and where the mean of the individuals corresponded to the bulk estimate, bulking was deemed to be satisfactory.

Total nitrogen estimates were carried out by the Biuret technique (Weichselbaum, 1946), and read in an E.E.L. photo-electric colorimeter using a 625 green filter. The total protein was obtained by multiplying the total nitrogen estimate by 6·25. Serum albumen was then estimated by precipitating the globulins with a 28·2 per cent. sodium sulphate solution at 37° C, and filtering through a Whatman 44 filter paper, and then estimating total nitrogen of the remaining albumen in the filtrate, photo-electrically as above (Howe, 1921). The sodium sulphate concentration of 28·2 per cent. was chosen as it gave good agreement with electrophoresis estimates although it is higher than Howe (1921) recommends.

Calves were weaned at 6 months so that the 6-month old group contain calves just weaned. Over the weaning period and up to 12 months of age, calves were fed 1 lb. of concentrates and acha hay (*Digitaria exilis*) in addition to grazing. This applies equally to both the wet and dry season. Milking cows were fed concentrates according to production throughout the year. During the dry season in addition the following supplementary feeding was given. From early January, 1959 until mid-April, 1959, growing

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A Case of Focal Symmetrical Encephalopathy in a Sheep.—*Concluded.*

metrical distribution of the lesions in the mid-brain suggested rather a different cause. The possibility cannot be overlooked that due to the sudden onset of neurological symptoms the animal could not maintain its balance and by falling against a wall caused the head injuries. On the other hand, consideration should be given to the striking similarity between this condition and focal symmetrical encephalomalacia in lambs described by Hartley (1956), Barlow (1958), and Robertson & Wilson (1958). The main difference was that in the lamb softening of brain tissue was invariably present, while in the above young adult sheep plaques of partial demyelination were found. The age incidence and to a certain extent the clinical signs bear resemblance to focal symmetrical spinal poliomalacia in sheep in Kenya (Plowright, 1954; Innes & Plowright, 1955), but while in the present case the lesions were confined to the brain, in the sheep in Kenya the spinal cord only was involved.

Acknowledgments.—The author wishes to thank Mr. J. C. Rennie for his technical assistance and also Mr. D. Watson for preparing the photomicrographs.

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TABLE I
COMPARATIVE AGE AND SEASONAL VARIATIONS IN SERUM PROTEIN FRACTIONS

Age group	July				October			
	Mean albumen g. per 100 ml.	Albumen standard deviation	Mean total protein g. per 100 ml.	Mean globulin g. per 100 ml.	Mean albumen g. per 100 ml.	Albumen standard deviation	Mean total protein g. per 100 ml.	Mean globulin g. per 100 ml.
0 to 2 months	2·87	0·5	5·34	2·81	2·81	—	7·13	4·72
6 months	2·98	0·26	6·90	3·92	2·40	0·39*	6·10	3·70
12 months	2·27	0·14	5·93	3·66	2·39	0·21*	6·42	4·03
18 months	2·01	0·41	6·34	4·33	2·20	—	7·11	4·91
2 years	2·33	0·27	6·61	4·28	2·25	—	7·11	4·86
2½ years	2·35	0·22	7·56	5·21	2·31	—	7·32	5·02
3 years	2·45	0·63	7·36	4·91	3·20	—	7·67	4·47
6 + years pregnant cows ...	2·43	0·50	8·59	6·16	2·00	—	8·21	6·21
6 + years non-pregnant cows	2·81	0·15	7·98	5·16	2·68	—	7·65	4·97
Total means	2·50	—	6·95	4·45	2·47	—	7·19	4·72

Age group	January				April			
	Mean albumen g. per 100 ml.	Albumen standard deviation	Mean total protein g. per 100 ml.	Mean globulin g. per 100 ml.	Mean albumen g. per 100 ml.	Albumen standard deviation	Mean total protein g. per 100 ml.	Mean globulin g. per 100 ml.
0 to 2 months	3·37	—	8·10	4·73	2·18	—	5·93	3·75
6 months	2·83	0·34*	5·76	2·93	1·74	0·22*	6· 1	4·36
12 months	3·14	0·39*	5·90	2·76	1·96	0·12*	5·88	3·92
18 months	2·44	—	6·79	4·35	1·98	—	5·74	3·76
2 years	2·81	—	7·33	4·52	1·80	—	5·10	3·30
2½ years	3·16	—	7·25	4·09	1·40	—	5·925	4·525
3 years	2·90	—	7·50	4·60	1·56	—	5·305	3·745
6 + years pregnant cows ...	2·62	—	8·77	6·15	1·90	—	7·25	5·35
6 + years non-pregnant cows	2·76	—	7·74	4·98	2·00	—	5·95	3·95
Total means	2·89	—	7·23	4·34	1·83	—	5·91	4·08

* Individual samples taken in 6 and 12 month groups only.

stock (12 months—3 year group) received maize silage at the rate of 5 lb. per head per day, and mature stock received maize silage at the rate of 10 lb. per head per day. From early March, 1959, until the end of April, 1959, growing stock also received concentrates at the rate of 1 lb. per head per day and acha hay *ad lib*. The constituents of the concentrates fed were guinea corn 2 parts, maize 1 part and ground nutcake 1 part.

Results

The estimates for serum protein fractions and serum albumen for the 4 seasonal observations on the 9 age groups are shown in Table I.

The rainfall figures for the year 1958 and the first quarter of 1959, are shown in Table II.

Serum albumen level differences (within observations) between age groups were not significant. There was a tendency, however, for a drop in serum albumen to occur between birth and 1 year of age. This drop was evident in all 4 seasonal observations although the differences were not significant. This may be an age drop or could be associated with changes in nutritional level associated with weaning. Another difference, consistent but not significant, was that between the pregnant and non-pregnant cow group. The "pregnant" cow group showed higher serum globulin levels while the "non-pregnant" cow group showed higher serum albumen levels.

A significant difference occurred in the 12-month groups between October and January. The rise in serum albumen which occurred in January in the 12-month group was fairly general in the other age groups. In January a wider variation in serum

(*Continued at foot of adjoining column*)

TABLE II
MONTHLY RAINFALL IN INCHES JANUARY 1958 TO APRIL 1959

Jan. 1958	Feb. 1958	March 1958	April 1958	May 1958	June 1958	July 1958	August 1958	Sept. 1958	Oct. 1958	Nov. 1958	Dec. 1958	Jan. 1959	Feb. 1959	March 1959	April 1959
0·38	0·57	0·72	7·83	4·83	7·76	6·95	5·83	6·11	1·14	Nil	Nil	Nil	Nil	r·67	2·89

An Osteodystrophy of Weaned Pigs

BY

E. J. JACK

Veterinary Investigation Centre, Starcross

NORMAL bone formation in the growing pig demands an adequate supply of calcium and phosphorus, a correct ratio between the two, and an adequate supply of vitamin D. The work of Theiler, du Toit, and Malan (1937) with growing pigs indicated that diets of either low calcium or low phosphorus in an abnormal ratio tended to have a more severe effect on bone formation than a diet deficient in both elements but of normal ratio. Morrison (1948) states that the dietary requirement of vitamin D for pigs varies in relation to the breed of pig (possibly due to the amount of skin pigment and its effect on ultra-violet light absorption), and to the amounts and ratio of calcium and phosphorus in the diet. Thus, when adequate quantities of both calcium and phosphorus are available in the correct ratio, rickets is unlikely to occur in white pigs (even bred away from direct sunlight) although the diet is almost devoid of vitamin D. The vitamin D content of the various grains fed to pigs is negligible. Deficiencies of other elements, *e.g.* manganese and fluorine, may lead also to incorrect bone formation.

This report describes a rickets-like condition without marked epiphyseal enlargement which occurred among weaned and fattening pigs in a closed Wessex Saddleback herd. The sows were housed out of doors during pregnancy, were farrowed mainly indoors, and occasionally outside in farrowing arks. The piglets were weaned at 8 weeks. Immediately after weaning the piglets went into the fattening house where their exposure to sunlight was very small. Approximately a fortnight after entering the fattening house, the pigs ceased to thrive and developed lameness.

The animals were fed dry with water *ad lib.* The affected pigs (weaning to fattening) received the following mixture: barley meal 1 part; weatings 1½ parts; flaked maize ¼ part.

As the ingredients were measured by volume and then mixed there was likely to be considerable variation in the above proportions. No supplements were fed. The figures for Ca and P and the protein contents were as follows:—

	CaO per cent.	P_2O_5 per cent.	Protein per cent.	CaO P_2O_5 ratio
Barley meal	0·07	0·84	—	—
Weatings	0·13	1·50	—	—
Flaked maize ...	Trace	0·60	—	—
Entire ration... ...	0·11	1·40	13	1:12·73
Recommended figures				
1. Weaners ...	1·00–1·50	1·40–1·60	16–18	1: 1·2
2. Fatteners ...	0·75–1·50	1·10–1·40	12–14	1: 1·1

It will be noted that the ration is deficient in calcium, has a very wide calcium-phosphorus ratio, and is deficient in proteins. Fish meal had been included in the diet of the affected pigs but was later omitted in favour of maize. It was from this time that the trouble started. Nursing sows received the above ration with the addition of 5 per cent. fish meal, while dry sows were fed on a home-ground dredge mixture of oats, barley, and wheat.

Lameness was the most striking feature of the condition. Animals were exceedingly reluctant to rise, and when forced to stand were in obvious and sometimes acute pain at the slightest movement. There was no obvious swelling of the joints or distortion of the facial bones; but very severely affected animals, particularly in the younger groups, had distinct bending of the limb bones. Two of 3 animals were unable to stand, and one of these had fractured a femur. Manual examination and palpation were

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Normal Serum Albumen Values in Nigerian Zebu Cattle.—*Concluded.*

albumen levels occurred. This variation and the general rise in serum albumen levels may be attributable to the effect of the supplementary feeding introduced early in January.

Significant differences occurred in both the 6-month and 12-month group serum albumen levels between January and April. Between these 2 observations a significant drop in the serum albumen levels occurred and this drop occurred in all age groups. At the time of the April examination, the grazing available was practically nil, and the herd, in particular the growing stock, was in relatively poor condition. This poor condition is reflected in the low mean serum albumen level, for all groups, of 1·83 g. per 100 ml. This and the mean serum albumen level of the 6 and 12 months groups in July and October of 2·51 g. per 100 ml. agrees with our previous figure of 1·9 g. per 100 ml. or less being an indication of pathogenic effect of haemonchosis in 6- to 12-month-old animals during the rainy season in Nigeria (Ross & Armour, 1959).

It can be concluded that these results substantiate previous findings that serum albumen level is a useful guide to condition.

Acknowledgments.—I am indebted to Dr. J. I. Taylor, Director of Veterinary Research, Mr. R. P. Lee, and Mr. J. Armour for their assistance during the preparation of the paper, and to Messrs. Shonekan and Enweani for technical assistance.

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difficult owing to the extreme distress caused. The most severely affected pigs were in the 3- to 5-month age group. After this age symptoms became less severe but the older pigs were exceedingly slow to fatten and had a hobbling lameness. It was quite likely that some of these older animals were weaned a few weeks before the fish meal was replaced by maize and this may have been responsible for their slightly improved condition.

FIG. 1.—An affected piglet showing severe stunting and bending of the limb bones.

FIG. 2.—A severely affected pig which was unable to stand.

A 3½-month-old neuter male and 4½-month-old female were examined *post mortem*. All bones of the carcases were exceedingly soft. With little effort it was possible to push a knife through the skull and long bones, and the ribs bent easily under pressure. There was no macroscopic enlargement of the epiphyses of the long bones or of the costo-chondral junctions; articular surfaces of all joints examined were macroscopically normal. The neuter male pig had a fractured humerus and several ribs showed large round swellings in their shafts. Histological examination of one of these lesions indicated that the swelling was due to curvature of the bone

(*Continued in adjoining column*)

Two Further Cases of Tetanus Treated with Chlorpromazine

BY

J. T. WICKENS and BRYAN BOSWOOD

Shenfield, Brentwood, Essex

Introduction

THE report by Evans (1959) of a case of tetanus in a dog stimulates us to record 2 subsequent cases of our own, one in a dog and one in a pony, both of which recovered.

Case Histories

Case One

A 3-month-old male Labrador puppy was presented to us on September 19th, 1959. It was reported that his face had quite suddenly become swollen, he was finding it difficult to eat, and his gait was somewhat stiff.

His temperature and respiratory rate were normal. He was able both to eat and to drink, though rather slowly. He could walk and run without difficulty, but his movements lacked the flexibility which characterises the gait of a normal puppy. His facial expression was certainly strange: it is possible that his lips were slightly swollen.

Tetanus was not suspected. A very tentative diagnosis of an allergic response to a possible wasp

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An Osteodystrophy of Weaned Pigs.—*Concluded.*

accompanied by osteoid deposition. All bones were thin.

Apart from severe stunting there were no other marked gross *post-mortem* findings and bacteriological examination showed nothing abnormal.

As immediate treatment the pigs were given daily supplements of calcium and phosphorus, and vitamins and suitable diets were compounded by the N.A.A.S. nutrition chemist for the various age groups of pig. Improvement was rapid and uneventful but the pigs remained stunted.

The farmer was asked to keep one pen of severely affected pigs as a control. By mistake these pigs were fed on a proprietary fattening meal. Although of excellent quality, this was not particularly rich in either calcium or phosphorus content, but even on this meal the pigs began to make a marked improvement.

Acknowledgments.—I wish to thank Mr. J. D. J. Harding (Weybridge) for histological examination, Mr. C. D. Rickaby (Starcross) for advice on nutrition, Messrs. Clements and Kay who took the photographs, and Mr. A. V. H. G. Abbott who kindly brought the case to our attention.

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sting was proposed and 25 mg. promethazine hydrochloride* were injected subcutaneously.

By the following day his condition had deteriorated. His gait was more obviously stiff; he was finding it so difficult to drink that it took him about 3 minutes to lap up a saucerful of milk. His tongue appeared to be swollen. Suspicion of tetanus was aroused, although it was impossible to provoke a tetanic spasm of any group of muscles at this stage. The possibility that a foreign body was causing the difficulty in eating and drinking was excluded by examination with X-rays and under general anaesthesia. Our impression was that on recovery from anaesthesia (intravenous thiopentone sodium) his stiffness was considerably more severe than formerly.

He was admitted to hospital kennels and kept away from other patients in a quiet dark room: 1,500 I.U. tetanus antiserum were given intravenously, and ½-gramme streptomycin, 250,000 units penicillin, and 50 mg. chlorpromazine hydrochloride† intramuscularly.

On the second day the stiffness was more marked and the diagnosis of tetanus was no longer in doubt; however, the animal was able to eat and drink provided that the food was put into his mouth.

He was given 3,000 I.U. antiserum subcutaneously, and ½-gramme streptomycin, 1 megaunit penicillin, and 50 mg. chlorpromazine hydrochloride intramuscularly. At night he was given 100 mg. chlorpromazine hydrochloride subcutaneously.

By the morning of the third day all 4 legs were quite rigidly stiff. He was unable to do more than lift his head an inch or two from the ground, and to wag his tail. He never lost control of his tail: we shall not forget the sound of the tail of this almost completely rigid dog, tapping against the ground whenever anyone came near him. He was unable to eat liver even if it was put into his mouth. His condition remained much the same for 4 days.

No further doses of antiserum were given on account of the risk of anaphylaxis, but penicillin and streptomycin therapy was maintained, not so much for its action against *Clostridium tetani* as to reduce the risk of hypostatic pneumonia. 100 mg. chlorpromazine hydrochloride were injected subcutaneously every 12 hours. A protein hydrolysate‡ was administered orally, with some difficulty, 3 times a day; between 100 and 200 c.c. of this hydrolysate were given daily.

On the seventh day it was noticed that he was swallowing more efficiently and that the movement of his head was stronger. On the eighth day he was able to lap again. From the eleventh day onwards he was able to eat meat.

Twice-daily injection of chlorpromazine hydrochloride was continued, but in gradually decreasing doses so that by the eleventh day he was receiving 50 mg. twice a day. Raw egg and milk were introduced into his diet on the eighth day and broth and minced meat on the eleventh day, the protein hydrolysate being gradually replaced by these foods.

On October 3rd, the fourteenth day of his illness, the puppy was able with assistance to walk very slowly and shakily round his kennel. The next day he got up and walked by himself. He continued to improve and on October 10th he was sent home.

In the middle of November he was vaccinated against distemper. He is a normal puppy.

Case Two

On October 23rd, 1959, a 9-year-old gelded pony was presented to us with a report that he had injured his back; he had fallen into a muddy ditch the previous day and since struggling out of it he appeared to walk abnormally.

His temperature was normal, but his pulse and respiratory rate were slightly increased. He both walked and trotted with his hind legs held rather stiffly and unusually far apart. His tail was very slightly raised. He had been seen to defaecate but not to urinate that day.

Three possible diagnoses were considered: urethral obstruction, inguinal lymphadenitis, and tetanus. To assist in differentiation ¼-gramme pethidine was injected intramuscularly and the pony was re-examined 3 hours later.

After this lapse of time there was no improvement. He did not resent vigorous palpation in the region of his groins. Borborygmi were audible and a rectal examination revealed normal faeces and an empty bladder. A tap under the jaw appeared to startle him—his head jerked backwards very suddenly—but no spasm of the nictitating membrane was detected. Repeated tapping failed to fatigue this reflex jerk. A tentative diagnosis of tetanus was proposed, and 10,000 I.U. tetanus antiserum were injected intravenously and 5,000 I.U. subcutaneously (this was all we had immediately available).

On the two days following there was still doubt in our minds with regard to the diagnosis, his condition remaining much the same. Nevertheless, 25,000 I.U. antiserum were injected subcutaneously on each day. 1 mg. carbamyl choline§ was also given intramuscularly the day after presentation as he had not defaecated; this injection appeared to cause him unusually great abdominal pain, and we now feel that the administration of this drug was a mistake.

By the third day his condition had deteriorated a little: stiffness was apparent in all his legs, and he was able to eat only if his food was raised 2 or 3 feet from the ground. Daily subcutaneous administration of 250,000 I.U. antiserum was continued.

On the fourth day he had difficulty in moving his head, and a typical tetanic spasm of the nictitating membrane was observed together with a spasm of the larynx causing a grunt; but he was still able to eat and drink food and water raised to his mouth.

At this stage 250 mg. chlorpromazine hydrochloride were injected intramuscularly.

On the fifth day he was able to lie down and get up, but his gait was no less stiff than formerly. We

* "Phenergan." May & Baker, Ltd.
† "Largactil." May & Baker, Ltd.
‡ "Protogest." B.W.

§ "Carbachol." May & Baker, Ltd.

A Note on Camels

We are indebted to Dr. N. S. Barron for this summary of a long article in the Scientific American, *written by Knut Schmidt-Nielsen.*

RECENT observations by several Americans on camels in the Sahara have added appreciably to our understanding of the proverbial ability of the camel to go without water for long periods. This does not appear to be due to any peculiarity of the stomach either anatomical or physiological, nor to the presence of a special water reservoir—often assumed to be under the hump. There is no special reservoir, nor does the camel lay in any extra reserves. Its ability to maintain itself under the most exacting conditions of heat and dryness depends mainly upon remarkable physiological factors.

In what is euphemistically described as winter in the Sahara camels are able to meet their needs for water by browsing on bushes and plants which have grown after the rains, even if no extra water is given to them. When deprived of this meagre source of moisture, however, they steadily lose weight due to water loss through the urine and the lungs.

When offered water camels drink only so much as will bring them back to their normal working weight—their saturation point so to speak. Deprive a camel of water for several days or compel it to go without for 3 weeks, which is about the maximum of its resistance, then it will drink avidly when water is offered. One camel was observed to take 27 gallons in 10 minutes which returned it to the weight it was before deprivation—and no more. The crux of the whole matter is the camel's ability to control its water and heat loss. Like the sheep the camel is able to restrict the amount and increase the concentration of its urine. It is also able to retain water by concentrating its urea and salt. The camel is known to favour bitter waters even though they may contain magnesium sulphate, and camels living on a seaboard will readily eat seaweed. Even efficient mechanism, however, cannot explain the camel's ability to survive on low water rations when temperatures reach 120°. Higher animals cannot survive much more than 100° F. unless they can seek shelter. In the case of the latter the thickened blood reaches a point where death occurs from "explosive heat," when the blood can no longer carry heat to the surface of the body to be lost. The camel avoids death by this means, by maintaining an even blood volume despite water loss from the tissues. Up to about 25 per cent. of the bodyweight can be lost in this way before the lethal limit is reached. One camel was observed to lose 220 lb. in 8 days (22 per cent. of its bodyweight). It shrank to a mere skeleton, but when offered water had no difficulty in rapidly recovering its normal appearance in about 10 minutes!

Whilst water is rigorously conserved, a certain amount is made available by oxidation of fat, a high proportion of which is stored under the hump. Thus 100 lb. of fat is equivalent to 110 lb. of water. The mechanism of heat loss plays an important part. There is practically no fat under the skin, the poor insulation aiding rapid heat loss. There is considerable matted hair several inches thick on the back where the sun beats down which protects the underlying tissues and reduces heat absorption. If deprived of this protective coat the animal produces 60 per cent. more sweat than a comparable unshorn animal. In excessive heat in the region of 150° F. the camel's temperature will rise slowly to 105° thus reducing sweating. When it reaches this stage it begins to sweat freely. During the cold night its temperature falls to as little as 93° F., there being a long delay before it is forced to its maximum—which is highly economical. Whilst man sweats

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Two Further Cases of Tetanus.—*Concluded.*

noted the coincidence of his regaining the ability to lie down and get up with the onset of chlorpromazine therapy.

Daily administration of antiserum and chlorpromazine was continued in the same dosage.

His condition remained unchanged until the ninth day, when he was again able to eat food from a bucket on the ground. He continued to improve and by the fifteenth day was walking normally.

Administration of chlorpromazine was discontinued on the eleventh day of the illness, and antiserum was discontinued on the thirteenth day.

Conclusion

Our impression is that chlorpromazine hydrochloride was of value in both these cases, not only by virtue of its sedation of patients likely to be alarmed by their own condition, but also by virtue of a partial relief of the muscular spasm itself.

It is interesting to note that at the height of their illness the puppy and the pony were receiving similar daily doses of chlorpromazine—200 mg. the puppy and 250 mg. the pony—despite the great difference in bodyweight. Nevertheless the pony was more markedly sedated than the puppy.

In neither case was there any noticeable loss of condition in spite of the difficulty of feeding.

In neither case was it possible to find signs of a recent wound through which *Clostridium tetani* could have gained entrance; but it was reported that the puppy had the habit of eating soil, and the pony had been shod some 10 to 14 days prior to the onset of symptoms.

We attribute the recovery of the puppy much more to the patient perseverance of our nursing staff than to specific therapeutic measures. We attribute the recovery of the pony to the early, if tentative, diagnosis, which probably resulted from our having the first case fresh in our minds.

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News and Comment

APPOINTMENT OF DEPUTY CHIEF VETERINARY OFFICERS

Mr. John Reid, M.R.C.V.S., D.V.S.M., and Mr. A. G. Beynon, M.R.C.V.S., D.V.S.M. (VICT.), have been appointed Deputy Chief Veterinary Officers of the Ministry.

Mr. John Reid will succeed Mr. A. D. J. Brennan, C.B.E., M.R.C.V.S., who will be retiring on March 31st, 1960, and Mr. Beynon will succeed Mr. L. Hughes, C.B.E., M.R.C.V.S., who will be retiring later this year.

AREA ERADICATION PLAN FOR TUBERCULOSIS

The final stage of the plan to eradicate bovine tuberculosis will commence on March 1st when those parts of England not already attested will be declared Tuberculosis Eradication Areas. These Areas, in which free tuberculin tests have been provided since March 1st, 1958, are:—

Northumberland and part of Durham; Cheshire, Derbyshire, Huntingdonshire, Isle of Ely, Leicestershire, Lincolnshire, Nottinghamshire, Rutland, Soke of Peterborough, Staffordshire, Yorkshire (East Riding), and parts of Lancashire, Northamptonshire, Warwickshire and Yorkshire (North and West Ridings).

From March 1st all herds in the Areas will be liable to compulsory tuberculin tests, and any reactors found will be slaughtered. Compensation will be at a rate of three-quarters of the market value of comparable attested animals subject to a maximum payment of £100.

THE PHYSIOLOGY SOCIETY

A meeting of this society was held at the Royal Veterinary College on Friday and Saturday, February 19th and 20th. There were over 26 demonstrations and 31 communications, under the chairmanship of Professor Amoroso or Dr. Jewell. Perhaps the highlight of the meeting was the closed-circuit colour television demonstration on Saturday morning. We believe that Mr. S. Jennings, Junior Vice-President of the B.V.A., is the first veterinary surgeon to appear on colour television in this country since he opened the demonstration by anaesthetising a mule and cow. He was followed by Professor Amoroso and Dr. J. N. McArthur who showed a versatile microscope for visualising histological material with colour television. Next, Mr. O. Graham-Jones demonstrated restraint and anaesthesia of a cage-bird, and finally Mr. E. W. Fisher provided a demonstration of the technique and practice of the clinical assessment of cardiac output in the horse by the dye-dilution technique.

The closed-circuit television was provided by Messrs. Smith, Kline, and French.

PROSECUTION BY THE R.C.V.S.

On January 27th, at the Liverpool City Magistrate's Court, The Royal College of Veterinary Surgeons successfully prosecuted Mr. George Wall, who was fined £10 and ordered to pay £5 5s. costs in respect of an offence under the Veterinary Surgeons Act, 1948.

TOXIC CHEMICALS IN AGRICULTURE

The Minister of Agriculture, together with the Minister for Science, the Secretary of State for Scotland, and the Minister of Health, have appointed 9 members to a research study group, under the chairmanship of the Chief Scientific Adviser on Agriculture to the Ministry, Professor Sanders.

The group's terms of reference are: "To study the need for further research into the effects of the use of toxic chemicals in agriculture and food storage, and to make recommendations."

This study group is independent of the interdepartmental Advisory Committee on Poisonous Substances used in Agriculture and Food Storage, which will continue.

The following are the names:—

Professor H. G. Sanders, Chief Scientific Adviser (Agriculture), Ministry of Agriculture; Dr. H. R. Barnell, Chief Scientific Adviser (Food), Ministry of Agriculture; Dr. J. M. Barnes, Director, Toxicology Research Unit, Medical Research Council; Professor A. R. Clapham, Professor of Botany, Sheffield University; Dr. R. A. E. Galley, Director, Tropical Products Institute, Colonial Office, Officer-in-Charge, Colonial Pesticides Research; Mr. C. O. Harvey, laboratory of the Government Chemist, Department of Scientific and Industrial Research; Dr. Donald Hunter, physician to London Hospital; Mr. W. C. Moore, Director, Plant Pathology Laboratory, Ministry of Agriculture; Professor W. L. M. Perry, Professor of Materia Medica, Edinburgh University; and Mr. G. G. Samuel, scientific staff (headquarters) Agricultural Research Council.

THE REGISTER OF VETERINARY SURGEONS

The name of Mr. Arthur Roland Bills, 8A, St. James Parade, Muswell Hill, London, N.10. was restored to the Register on February 19th, 1960, under the provisions of Section 25 (4) of the Veterinary Surgeons Act and S.I.1729 of 1957.

A Note on Camels.—*Concluded.*

from sunrise to sunset a camel limits his sweating time to a minimum. The latter tolerates a far greater degree of dehydration, and loses water better and more slowly than man.

Post-mortem examinations failed to reveal any special water storage organ. Though there are extra pouches in the rumen the total additional capacity of these is not more than ¼ gallon and the contents resemble those of the rest of the organ. All stomachs, however, contain considerable fluid, but less than the stomachs of other ruminating animals. No organs or tissues appeared to be especially dehydrated. Fat was concentrated under the hump.

Donkeys were observed to tolerate about 25 per cent. reduction in bodyweight due to water loss, this process taking place three times as fast as in the camel. Whilst the latter will survive for from 17 to 21 days without drinking, the donkey under test had to be watered once every 4 days. Its body temperature was far more stable, fluctuating very slightly. Whilst the camel requires 10 minutes to restore its tissues to their full strength, any loss in this respect can be replenished by the donkey in 2 minutes.

THE COLONIAL VETERINARY SERVICE

Promotions. D. L. Kelly, M.R.C.V.S., D.V.S.M., (ex-Bechuanaland Protectorate), Veterinary Officer, Tanganyika, becomes Senior Veterinary Officer, Tanganyika; J. P. J. Ross, M.R.C.V.S., D.T.V.M., Veterinary Officer, Tanganyika, becomes Senior Veterinary Officer, Tanganyika.

New appointments. C. W. Brown, B.SC., M.R.C.V.S., Veterinary Officer, Bermuda (has served in Kenya); A. D. Morris, M.R.C.V.S., Veterinary Officer, Uganda (has served in Ibadan, Western Nigeria); D. Shannon, B.SC., M.R.C.V.S., Veterinary Officer, Kenya (resigned but re-employed); K. S. Thomson, M.R.C.V.S., Veterinary Officer, Tanganyika (resigned but re-employed); and D. Robertshaw, M.R.C.V.S., Scientific Officer (physiologist), E.A.V.R.O., Muguga, Kenya.

COMING EVENTS

March

1st (Tues.). 98th Annual General Meeting of the Lancashire Veterinary Association at the Veterinary Hospital, University of Liverpool, 2.30 p.m.

2nd (Wed.). Annual General Meeting of the Scottish Metropolitan Division in the Royal (Dick) School of Veterinary Studies, 2.30 p.m.

Annual General Meeting of the A.V.T. & R.W. Northern Region at the Veterinary Hospital, Pembroke Gardens, Liverpool, 1.45 p.m.

Annual General Meeting of the South Wales Division at the Ivy Bush Hotel, Carmarthen, 2.15 p.m.

A Meeting of the South Wales Ladies' Guild will be held in the lounge of the Ivy Bush Hotel (see notice above), 2.30 p.m.

3rd (Thurs.). Meeting of the Society for the Study of Animal Breeding at the Royal Society of Medicine, 1, Wimpole Street, London, W.1, 11.30 a.m.

Glasgow University Veterinary Medical Association Annual Ball in the St. Enoch Hotel, 8 p.m. to 2 a.m.

Meeting of the Central Veterinary Society at the Royal Veterinary College, 6 p.m.

Meeting of the B.S.A.V.A. Midland Region at The Oaks Hotel, Shelton, Shrewsbury, 8 p.m.

7th (Mon.). Meeting of the Yorkshire Division in the Talbot Hotel, Malton, 8 p.m.

9th (Wed.). Meeting of the Southern Counties Veterinary Society at the Royal Hotel, Winchester, 7.30 p.m.

General Meeting of the South-East Midlands Veterinary Association at Franklins Gardens Hotel, Northampton, 7.30 p.m.

Meeting of the Staffordshire and Derbyshire Veterinary Club at the White Hart, Uttoxeter, 7.30 p.m.

10th (Thurs.). Meeting of the Essex Veterinary Society at the Essex Institute of Agriculture, Writtle, 7.30 p.m.

11th (Fri.). Meeting of the Midland Counties Veterinary Association at the Birmingham Medical Institute, 36, Harborne Road, Birmingham, 7.30 p.m.

16th (Wed.). Meeting of the North Wales Division at the School of Agriculture, University College of North Wales, 2 p.m.

Meeting of the R.S.M. Section of Comparative Medicine at 1, Wimpole Street, London, W.1, 5 p.m.

23rd (Wed.). General Meeting of the Lancashire Veterinary Association in Preston, 2.30 p.m.

Spring Meeting of the Society of Women Veterinary Surgeons at The Priory, Thurgarton, 2.30 p.m.

April

2nd (Sat.). 14th Annual Dinner of the B.R.X. Club at the Royal Veterinary College, London, N.W.1, 6 p.m.

7th (Thurs.). 12th Meeting of the British Veterinary Poultry Association at the Royal Veterinary College, Camden Town, N.W.1, 11 a.m.

9th (Sat.). Symposium of the Forensic Science Society at Guy's Hospital Medical School.

30th (Sat.). Meeting of the N.W. Region of the B.S.A.V.A. at the Queen's Hotel, Manchester, 7.30 p.m.

August 28th to September 3rd. 78th Annual Congress of the British Veterinary Association in Glasgow.

B.V.A. COMMITTEE AND COUNCIL MEETINGS IN EDINBURGH

Tuesday, April 19th
Late Evening. Finance Sub-Committee.

Wednesday, April 20th
10 a.m. Small Animals Committee.
12 noon. Home Appointments Committee.
2.15 p.m. Veterinary State Medicine Committee.
4 p.m. Parliamentary and Public Relations Committee.

Thursday, April 21st
10.30 a.m. Farm Livestock Committee.
2 p.m. General Purposes and Finance Committee.

Friday, April 22nd
10.15 a.m. Council Meeting.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Ches. Lane End Farm, Ashton, Chester (Feb. 16); Moss Farm, Bickley, Malpas (Feb. 17).
Cumbs. Hill End, Armathwaite, Carlisle (Feb. 18).

Fowl Pest

Berks. Wartah Poultry Farm, Gorse Drive, 9 Mile Ride, Wokingham (Feb. 18).
Cambs. 58, Cam Causeway, Chesterton (Feb. 19).
Essex. 458, Rayleigh Road, Thundersley; Hall Farm, Radwinter, Saffron Walden; Maori, Berwicks Road, Rainham (Feb. 22).
Hants. Stonycroft, Grosvenor Road, Soldridge, Medstead, Alton (Feb. 16); 50, Corbin Road, Pennington, Lymington;

North Lodge, Longstock Park, Stockbridge (Feb. 17); The Duckery, Leckford Estate, Stockbridge (Feb. 20).

Kent. Tubbs Farm, Leigh Green, Appledore Road, Tenterden (Feb. 16); Hillborough Farm, Herne Bay; Whitehall Farm, Lydd, Ashford (Feb. 18); Kandy, Sweechbridge Road, Hillborough, Herne Bay; Fartyacre Farm, Lydd, Ashford; Churchmans Farm, Kennawayn, Faversham; Leigh Green Fruit Farm, Tenterden (Feb. 19); Danemore Lands Farm, Speldhurst, Tunbridge Wells (Feb. 22).

Lincs. Boston Road, Horncastle; 55, Tennyson Gardens, Horncastle; Southfield Place, Horncastle; Southfield Piggeries, Boston Road, Horncastle (Feb. 16); Brackenborough Road Nurseries, Louth (Feb. 18); The Cottage, Low Road, Wyberton, Boston (Feb. 21); 46, Brackenborough Road, Louth; Mount Pleasant, Gedney Dyke, Spalding (Feb. 22).

Middlesex. Hithermoor Farm, Stanwell Moor, Staines (Feb. 18).

Norfolk. 6, The Row, Scottow, Norwich; Holly House, Briston, Melton Constable; Primrose Farm, Bunwell Street, Bunwell, Norwich; Pond Farm, Bunwell, Norwich; Ferry Road, Norton Hales, Norwich; Northwold, Thetford (Feb. 16); 2, Nursery Cottages, Lyndford Cross; Mundford, Thetford (Feb. 17); Uttings Farm, Pringland, Norwich; Norwich Road, Denton, Harleston (Feb. 18); The Street, Marlaske, Aldborough; Grove Farm, Southburgh, Thetford; Bristows Farm, East Ruston, Stalham, Norwich (Feb. 19); Tottington Lane, Roydon, Diss; Rectory Cottage, Wickmere, Norwich; Pond Farm, Southburgh, Thetford (Feb. 20); Nr. Post Office, Moulton St. Mary, Norwich (Feb. 21); Brick Kiln Farm, Garvestone, Norwich; The Gables, Bawdeswell, Dereham; Beech Road, Hemsby, Great Yarmouth (Feb. 22).

Suffolk. 32, Roughlands, Lakenheath, Brandon (Feb. 16); Broom Corner, Lakenheath, Brandon (Feb. 18); Drybrook, Undley Road, Lakenheath, Brandon (Feb. 19); Rosman, Broom Road, Lakenheath, Brandon (Feb. 22).

Surrey. Rose Lea, North Looe, Ewell, Epsom; 135, Reigate Road, Ewell, Epsom (Feb. 18); Wyvenhoe, Guildford Road, Great Bookham, Leatherhead (Feb. 19).

Sussex. Shortgate Farm, Shortgate, Lewes; Belle View, Shortgate, Lewes; Beacon View, Shortgate, Lewes (Feb. 16); Burnt House Farm, Burwash, Etchingham; Marstella, Ruslake Green, Heathfield; Hornicks, Ruslake Green, Heathfield; Uplands, Fyfie Lane, Etchingham (Feb. 20); The Rectory, Burwash, Etchingham (Feb. 21); Court Farm, Burwash, Etchingham (Feb. 22).

Yorks. Railwayside, Cross Road, Keighley (Feb. 16); Gillmoor Farm, Bishop Thornton, Ripley, Harrogate (Feb. 18); Swillington Common Farm, Halton, Leeds, 15 (Feb. 21).

Swine Fever

Bucks. Slade Farm, Hedgerley, Slough (Feb. 16).

Cornwall. Cambrose, Nancekuke, Illogan, Redruth (Feb. 22).

Derbys. Hollybush Farm, Crich, Matlock (Feb. 17).

Dorset. Clenston Farm, Winterborne Clenston, Blandford Forum (Feb. 18); Nylon Farm, Kington Magna, Gillingham (Feb. 22).

Hants. Woodcote, 38, Reading Road, Chineham, Basingstoke (Feb. 18).

Kent. Pratts Farm, Hilden Park Road, Hildenborough (Feb. 18).

Lancs. Oak Farm, Oak Lane, West Derby, Liverpool (Feb. 22).

Norfolk. Old Hill, Hall Lane, Drayton, Norwich (Feb. 17).

Northamptonshire. Rectory Farm, Ecton (Feb. 18).

Oxon. Harris' Gardens, Bicester; The Croft, Hook Norton, Banbury (Feb. 22).

Staffs. Emergency Slaughterhouse, North Street, Brierley Hill (Feb. 17).

Somerset. Lower Riadge Farm, East Compton; Hengrove Farm, Hengrove Lane, Bristol, 4 (Feb. 16); Foxlands Poultry Farm, Walton, Street (Feb. 22).

Suffolk. Pit Farm, Badwell Ash, Bury St. Edmunds (Feb. 16).

Warwicks. Barnwells Barn Farm, Thurlaston, Rugby (Feb. 22).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Analyses of " Magnesium-rich " Mineral Mixtures

Sir,—Dr. Watt's letter in THE VETERINARY RECORD, February 6th, indicates only too clearly the disturbing ignorance of *some* manufacturers of magnesium-rich mineral supplements. Fortunately for all concerned there are other firms whose hypomagnesaemia-preventive mixtures display much higher magnesium levels as well as closer calcium to phosphorus ratios than the 6 instances described by Dr. Watt. To illustrate this, I give below the percentage magnesium, calcium and phosphorus content of several nationally known products now available.

I am sure your readers will agree that these are very much more rationally based than the heterogenous collection Dr. Watt has encountered.

Dr. Watt also mentions that "the mere publication of a formula is insufficient as the farmer cannot be expected to know the recommended amounts of magnesium to be fed." It has been the purpose of many of the more reputable mineral supplement manufacturers to attempt to educate the farmer as to the correct level of minerals to add to his livestock diets and, in fact, such firms have spent a good deal of money to this effect in literature, lectures and technical communications.

Product	Ca : P ratio	Declared analysis (Ca, P, Mg only)			Amounts of Ca, P, Mg (grammes) per lb. concentrates when added as directed by makers			Amount of mineralised concentrates necessary to acquire a daily intake of 30g. per mg.
		Ca	P	Mg	Ca	P	Mg	
A	1·5 : 1	5·3%	3·5%	35%	0·5	0·3	2·8	10½ lb.
B	3·37 : 1	15.2%	4·5%	13·5%	3·7–4·5	1·1–1·4	3·3–4·5	7–9 lb.
C	2·23 : 1	8·6%	3·7%	27%	1·4–1·8	0·6–0·9	4·4–6·6	4½–7 lb.
D	3·4 : 1	15·3%	4·5%	15%	1·9	0·6	1·8	16 lb.

Finally, may I make a plea for the wider use of an innovation which we instituted several years ago and which would allow the farmer's technical adviser more easily to estimate the contribution that any mineral supplement is likely to make to the diet. This is, in effect, to publish *two* formulae with each product. The first detailing the percentage of the various elements present in the bag; the second (*and far more important*) is to publish the amounts of the elements the animal would ingest daily (expressed as grammes or milligrammes per pound of concentrate feed) when the mineral supplement is added at its recommended rate.

An example of this technique in use, is to be found in the Table above and serves to indicate that *both* formula *and* rate of use require to be known to make a proper assessment of the value of any supplement.

Yours faithfully,
J. N. GADD,
Nutrition Adviser.

Boots Pure Drug Co. Ltd.,
Station Street,
Nottingham.
February 18th, 1960.

Virus Disease in Wild Foxes

Sir,—In our previous letter we promised a further communication if, and when, the position was clarified.

This is a progress report on the situation as we see it. So far we have received foxes from the following counties: Northamptonshire, Cambridgeshire, Leicestershire, Essex, East Essex, Bedfordshire, Sussex, Oxfordshire, Worcestershire, Hertfordshire, Gloucestershire, Surrey, Yorkshire, Northumberland, and Lincolnshire.

Chemical analyses on numbers of foxes received for *post mortem* have been negative for the following: mercury, arsenic, lead, zinc, phosphorus, fluorides, alkaloids, and warfarin. In two cases strychnine was found. In addition to the preceding list other chemical tests have been carried out for Antu rat poison and the various hydrocarbons used as agricultural insecticides. Results have, been negative.

The salient *post-mortem* lesions have been those associated with an acute disease, with haemorrhages throughout various organs but mainly in the brain, heart, lungs, and intestines. The livers have shown considerable variation, from centri-lobular necrosis to mottling and severe congestion. The most common finding has been mucoid blood-stained extravasations throughout the length of the bowel—sometimes dark and tarry in appearance. An interesting feature has been that this has been so acute that the animal has not even developed diarrhoea and the brush and anal region have not been stained. The majority of these cases have had sterile heart blood, and no pathogenic bacteria have been isolated.

On January 8th, 1960, we transmitted brain, liver, and spleen suspensions from a fox into 2 nine-week-old greyhound puppies which were negative to the complement fixation test for canine virus hepatitis immediately before inoculation.

On the third day they both developed bloody diarrhoeas, were off their food, had a tucked-up appearance, objected to abdominal palpation, showed a rapid loss of condition, and one animal had a scleritis. Both these puppies slowly recovered.

Material from another fox was passaged into a third puppy with similar clinical manifestations. It is intended to follow up serology on these cases.

Brain and liver from the donor foxes used as an antigen gave a positive complement fixation test against virus hepatitis serum of canine origin; we think this very significant.

The position with regard to inclusion bodies is not yet clear. In the majority of fresh carcases we have failed to find these, but in a few, atypical inclusion bodies have been observed.

From the foregoing summary we have little or no doubt that this disease is a virus infection very similar to fox encephalitis as described by Green (1930) in the U.S.A.

The complement fixation test has, to a great extent, indicated the serological identity of this virus with canine virus hepatitis and we are wondering whether the affinity of the disease in foxes and dogs may not be closer than is generally thought in this country. Several reports have been received of virus hepatitis in hounds and we have been able to conduct some *post-mortem* examinations on foxhounds where lesions were very extensive, particularly haemorrhages in the bowel and round the roots of the optic nerves and right cerebrum—so much so that we suspected a possibility either of leptospirosis or warfarin poisoning. Both these were subsequently negatived. Rather to our surprise inclusion bodies were found in liver cells and some carcases have been jaundiced. While jaundice is not common in canine virus hepatitis we have had such cases in foxes.

The M.F.H. Association has, naturally, taken a very keen interest in this problem and we have been officially asked to do all we can to clarify the position. Since we made our first observations many other laboratories have become involved and interested, and to assist us we should welcome any collaboration they are willing to give which, of course, would be reciprocated.

Yours faithfully,
S. F. J. HODGMAN,
R. S. T. BOWDEN.

Canine Health Centre,
Kennett,
Nr. Newmarket,
Suffolk.
February 15th, 1960.

References

GREEN, R. G., ZIEGBER, N. R., GREEN, B. B., & DEWEY, E. T. (1930). *Amer. J. Hyg.* **12**. 109.
HODGMAN, S. F. J., & BOWDEN, R. S. T. (1960) *Vet. Rec.* **72**. 54.

[Owing to postal delay this letter arrived after last week's issue had gone to press.—*Editor.*]

A Purchase Tax Warning

Sir,—Further to your published correspondence on "Dictol," your readers may be interested to hear that on receipt of the maker's letter stating that because the vaccine is given orally it attracts purchase tax, I despatched the following telegram to the Minister of Agriculture: "Request your immediate intervention to-day's fantastic imposition of purchase tax cattle husk vaccine. Reply paid and awaited with interest." I did this in my private capacity as a Government supporter interested in the elimination of bureaucratic nonsense in whatever form it may appear. As after a fortnight I had not received a reply I then saw my M.P. and informed him of the position. Two days later I received the following reply:—

Ministry of Agriculture, Fisheries and Food,
Whitehall Place,
London, S.W.1.
February 16th, 1960.

Dear Sir,

I am replying on behalf of the Minister to your telegram about purchase tax on the new oral vaccine "Dictol" for the prevention of husk.

The Government, after careful consideration, have reached the conclusion that there is as yet insufficient knowledge of its efficacy under general field conditions to justify exemption from purchase tax. I enclose your reply-paid voucher.

Yours faithfully,
Jean Arthur,
Private Secretary.

It would appear that it is up to the makers and the members of our profession to provide sufficient proof of the vaccine's efficacy to persuade the Government to improve its outlook.

Yours faithfully,
J. B. EDWARDS.

1, London Road,
Wellingborough,
Northants.
February 22nd, 1960.

The Clinical Specialist

Sir,—The article, "Whither Veterinary Education?" by Dr. J. T. Abrams (*Vet. Rec.* **72.** 123), disagrees with some of my views on the development of veterinary medicine. Dr. Abrams's laboratory is about 30 yards from mine and it might seem unnecessary, therefore, for us to debate our opinions in your columns. The only excuse for doing this must be that, in pursuing our differences in this way, we might clarify our arguments for others.

I have studied all the main points made by Dr. Abrams and find that most of these have already been covered in my two articles on clinical medicine (Goodwin, 1959a, b). There, I have commented on the tendency for the development of larger units in agriculture, the demand for advice to prevent rather than to cure disease, the enormous growth in knowledge relating to veterinary science, and the consequent difficulties facing general practitioners. I had not touched upon Dr. Abrams's further remarks about the earlier isolation of the profession, however, as this does not appear to be an issue among any members of the profession below about 50 years of age known to me.

We are, then, left only with the question of specialisation in the clinical field. Here, Dr. Abrams appears to have misunderstood my proposals, probably because he is not engaged in clinical medicine himself. When I advocate the development of species specialists, I am taking but a first step in sharing the burden and improving the quality of teaching and consultant advice at the clinical level. I have admitted that other forms of specialisation have their place, but problems in the field arrive as species ones first and must be dealt with by somebody who can handle them as such. Of course there are similarities between the biochemistry of pigs and dogs; but there are likewise parallels between the metabolism of dogs and men. Yet we have separate clinicians for human disease, and it is just as logical to have separate clinicians for other species. Somebody has to ensure that the wood is not obscured by the trees, and this is the task of the clinician. I have never pretended that the wood could exist without the trees; on the contrary, I have stressed repeatedly the indispensable contribution of other disciplines to medicine. I continue to disagree with Dr. Abrams, therefore, when he says that species specialisation is a retrograde step. I have tried both types of specialisation in consultant practice and was forced to admit several years ago that most disease problems in medicine demand a species specialist in the first instance.

When I defined my species specialist, I did not say that he would be an expert in the physiology, nutrition, housing, and so on, of one species. All I argued was that the modern clinician, especially if he is dealing with species like poultry or pigs, must be aware of "the behaviour of disease in relation to housing, nutrition, and management." Even if more non-veterinarians enter clinical departments, which I am not conceding to be desirable at this stage, this does not alter the fact that somebody has to examine the patient or the problem first and later correlate all the relevant facts. The alternative is a clinical circus that travels about the country and contains a member of every discipline that might be required.

It seems to me that Dr. Abrams's view cannot be reconciled with the practical demands either of the client or a clinical department. An experienced species specialist can deal with many diagnostic problems and give advice on corrective management without seeking the help of others. In most of the remaining instances he will know to whom he should go for assistance. He may have to try a number of more specialised advisers but only he can keep the case history integrated. Again, in teaching medicine, and most of our students will become general practitioners, one of the main needs is for instruction in differential diagnosis. Only a man experienced in all types of disease in a species can illustrate the complexities of differentiating similar clinical syndromes.

In conclusion, I am glad to have this opportunity of returning to the subject of preventive medicine. Dr. Abrams speaks of preventive medicine and

clinical medicine. I recognise no such distinction. As a clinician, much of my work is in the preventive field and I find that I am limited in this work largely by my knowledge of the pathogenesis and epidemiology of any particular disease problem. The idea that preventive medicine is a subject separate from clinical medicine is dangerous and has already gone too far. Studies in physiology, nutrition, etc., are vital to our understanding of the normal animal, but they do not together comprise preventive medicine. If a clinical teacher ignores preventive medicine, he will be a less adequate teacher; he will be little better off, however, if he is content to let a person untrained in disease try to teach the subject for him.

Yours faithfully,
R. F. W. GOODWIN.

School of Veterinary Medicine,
Madingley Road,
Cambridge.
February 20th, 1960.

References

GOODWIN, R. F. W. (1959a). *Vet. Rec.* **71**. 674.
———. (1959b). *Ibid.* **71**. 879.

" Progress "

Sir,—Mr. Hanscomb's letter in your issue of the 20th instant naturally interested me very much, but I would have expected his acknowledgment to have been to the increase in canine vaccination which he accepts as having arisen through pharmaceutical houses drawing the attention of the public to the possibilities of immunisation against the disease to which the canine species is heir. Since veterinary surgeons cannot undertake this task by personal advertisement, and since the profession has always found it difficult to advertise its services, a large number of veterinary surgeons in practice have welcomed the action of the pharmaceutical houses.

Obviously such advertising cannot be other than linked to a particular brand, but it seems to me that this disadvantage must be accepted if the main advantage is wanted and desirable. These prophylactics being restricted in their distribution to the profession is an insurance that the practitioner has the opportunity properly to inform his client according to the view which he has developed as to the suitability of any particular brand of product and as to the safety and quality of response which, on his assessment, can be expected. True it is, I imagine, that some clients will attempt to demand that their dog be immunised in a particular way with a particular product; this may arise as an effect of pharmaceutical house advertising, or on the basis of the client's experience or on dog world tittle-tattle. In any circumstances the veterinary surgeon will surely be the adviser and guide of his client.

Mr. Hanscomb objects to some of the points which are stressed in this lay advertising, but I would have thought the term " lasting immunity " a proper one to use in the present state of our knowledge and just the type of statement which one would expect the veterinary surgeon to interpret to his client, according to his views. The pharmaceutical industry in Britain has given the profession a high level of support in the work it has done in the development and provision of canine vaccines. That one set of research workers in this field should reach a different conclusion from another is only to be expected. They work in the field of biology and therefore one cannot expect the precision which is characteristic of the chemical sphere, where reaction and outcome are so much more certain.

Mr. Hanscomb made comment on the variety of recommendations for use which exist at the moment. It is in these comments that I am particularly interested. Conscious of the need to discuss the basis of some of these recommendations 2 of my colleagues have the preparation of an informative article almost completed. Pending its appearance, I feel I need only comment on some of the points of principle which Mr. Hanscomb has raised.

We have not seen in the laboratory, or have been able to obtain from the field, the evidence which would support Mr. Hanscomb's belief from his clinical observation that the immunological response to egg-adapted distemper vaccination is less good or less prolonged than that obtained by the old methods which depended for their success on the injection of living virulent virus. When the old methods were in use we had instances of apparent failure to immunise as we have to-day. Unfortunately the dog is not a standard animal; its response to the injection of an immunising agent varies from individual to individual, and even in the case of the individual from time to time. There is evidence that if the dog can respond, the response is not materially different whether egg-adapted virus or virulent virus is injected. Should it respond, the humoral immunity declines in much the same variable way no matter whether it follows exposure to virulent virus or the injection of an egg-adapted living vaccine. Unless maternal immunity or acquired immunity has fallen sufficiently low there is no antigenic stimulation from exposure to natural infection or to vaccine injection. One cannot expect that every exposure to natural virulent virus does boost immunity; one must accept that the widespread use of vaccine will reduce the opportunity for challenge at the time or times when the dog could respond. This point has not been lost sight of by research workers and observations are on hand which will provide some relatively clear, but it can only be general, definition of the position. It is because of this somewhat fluid position that we do not accept the term " lasting immunity " as misleading, as was suggested.

Since hepatitis vaccine is a relatively recent introduction it is not surprising that Mr. Hanscomb was particularly concerned with different types of vaccines which are available.

Whether a combined distemper/hard-pad/hepatitis vaccine is offered, or the hepatitis component provided separately, depends very largely on the type of vaccine used to convey a hepatitis immunity. With a living tissue culture-adapted hepatitis virus there are no good immunological grounds for keeping the 2 types of vaccine separate. The combining of them is based on evidence that this can be done satisfactorily to the convenience of the veterinary

surgeon, the dog owner and the dog itself. It is not a case of business expediency.

With regard to the age at which hepatitis immunisation should be practised, there is room for different recommendations depending on the circumstances of the case. It is the function of the veterinary surgeon in practice to consider all the circumstances in which he is invited to advise or act, and to determine from his knowledge and experience what is best. It is only after careful study of the available scientific evidence and consultation with veterinary surgeons in practice that recommendations can be made. It is better that a general one should be made, in view of the variation in the age at which puppies become susceptible and able to respond to the stimulus of the injection or vaccine. It is true that hepatitis tends to occur in puppies of a younger age than that at which distemper/hard-pad disease can be expected. This appears to be only in part associated with the disappearance of maternal immunity. It is, perhaps, more closely associated with exposure to infection—from the carrier animal—at an earlier age. It is therefore realistic that the need for, and an opportunity to, immunise at an earlier age than 9 weeks be pointed out and a suitable product provided.

Mr. Hanscomb is, apparently, not entirely satisfied that pig tissue culture-adapted living hepatitis vaccine virus is completely safe, fearing that it may regain its virulence as a result of accidental serial passage. It is known that this virus may pass from the inoculated dog to its contacts. Several attempts have been made by such serial passage to encourage it to regain its virulence. None have succeeded.

So far as leptospirosis is concerned the provision of the 2 types of vaccine separately has a historical background and a certain level of justification in the belief that immunisation against *L. icterohaemorrhagiae* had best be effected at an earlier age than that for *L. canicola*. There are no good immunological grounds why these 2 vaccines should not be combined; the combination, however, does give the veterinary surgeon a tool which is more easily applied.

I am sure that no one in the profession would do other than welcome further research in this field to cover the points which Mr. Hanscomb mentioned and the many others which await elucidation. Perhaps the contribution which pharmaceutical organisations have made to obtain the answers now available will only be fully appreciated by the profession when it realises the amount of money which would have to be raised to cover the programme Mr. Hanscomb outlined.

Yours faithfully,
R. F. MONTGOMERIE.

The Wellcome Research Laboratories,
Beckenham,
Kent.
February 22nd, 1960.

Sir,—I find myself in such violent disagreement with all the views expressed by Mr. Vaughan that I sincerely hope they are only the views of a small minority.

I am amazed that even with such a formidable and complex problem as mastitis he should condone a non-scientific and empirical approach.

It is said that the criterion of a useful professional life is that we leave the status of our profession a little higher than we found it. Mr. Vaughan may leave himself a lot to redress in the future.

Yours faithfully,
W. GRIFFITH JONES.

The Surgery,
"Oakfield,"
Atherstone.
February 16th, 1960.

Sir,—I would like to make a few comments in reply to Mr. Hanscomb's letter in your last issue. Undoubtedly there is a great deal of truth in what he says. It is unfortunate that in order to attract the interest of the public and to stimulate them to ask for products from veterinary surgeons, certain claims have to be made. I think that in most cases the statements are justifiable when made to the public, but are not sufficiently comprehensive from a professional point of view. It is our feeling that the veterinary surgeon is not adequately instructed in the use and storage of such products.

The egg-adapted living vaccine for the distemper/hard pad complex is undoubtedly the most satisfactory vaccine available. Your correspondent, however, suggests that clinical observation leads him to believe that the individual response is never as good, nor as long lasting, as the old vaccines using a field strain, and that the egg-adapted vaccine requires an early challenge in order to enhance the dog's immunity. This is in our opinion not altogether true, and we feel that prolonged immunity can be stimulated by use of an egg-adapted living vaccine, but in order to do so the vaccine must be properly prepared and properly handled after preparation. In the tests carried out with our vaccine the level of immunity has been found to be high in older animals not exposed to challenge early in life.

It has been demonstrated that the viability of lyophilised virus varies to a great extent according to methods of preparation and storage. It is, in our opinion, absolutely essential that all such vaccines should be maintained under refrigerated conditions for the duration of their shelf life, and we are satisfied that if this process is adopted it is possible to maintain the viability of the vaccine, thus enabling a high level of immunity to be established. An egg-adapted vaccine is more likely to fail through faulty procedure than through the inability of the material to set up a high level of immunity.

No one, of course, can claim that the immunity to distemper subsequent to vaccination would be life-long without challenge by contact with infected animals, but the necessity for early challenge is not quite so urgent as your correspondent would indicate.

There is undoubtedly a case for the vaccination of puppies with live hepatitis vaccine early in life, but I think your correspondent in his letter puts forward a fallacious argument in accepting the tacit assump-

tion that hepatitis is a disease only of puppies. The idea that dogs are either immune or dead as a result of hepatitis at 9 weeks is surely inaccurate. Many dogs avoid infection early in life but succumb to hepatitis later. The increasing trend to vaccination against hepatitis is reducing the weight of infection and allowing some dogs to reach maturity without exposure, thus creating a susceptible dog population. This explains why more and more dogs are suffering from hepatitis much later in life than ever before. For this reason it is becoming increasingly important to vaccinate against hepatitis as routine in order to protect the dog in later life. Surely the most convenient time to do this is when the dog is being vaccinated against distemper. It has been proved by use that live hepatitis vaccines are completely safe. It is safe to assume that if this virus was going to regain its virulence as a result of serial passage, it would have done so before now.

It is only as a result of extensive and painstaking tests that the claim is made that such a vaccine is fully avirulent and cannot reproduce the disease.

Yours faithfully,
T. STEWART.

Bayer Products Ltd.,
Neville House,
Eden Street,
Kingston-upon-Thames,
Surrey.

February 22nd, 1960.

Sir,—In reply to the interesting letter from Mr. Hanscomb, the lay press advertising of this laboratory, which began only recently, is in response to the direct request of many veterinary surgeons.

Distemper Complex. The method of immunisation prior to the introduction of egg-adapted virus in 1953 failed to produce a satisfactory immunity; hence, breakdown of immunity led to the use of the term " hard pad " as a separate clinical entity to distemper. My work on the strain " Glasgow S123 " of distemper established the possibility of producing a variety of clinical syndromes (including " hard pad ") by using suitable route of infection (Mansi, 1951). This work, I am glad to say, has been supported by various workers throughout the world during the last 9 years (Cabasso, 1952).

Although I am inclined to agree that immunity produced by egg-adapted distemper virus is not equal to that produced by a natural virus, this vaccine followed by chance exposure to field infection will provoke a life-long immunity. This point was fully dealt with in the introductory pamphlet on " Weyvak " distemper complex vaccine issued to the veterinary profession.

Over-adaption of the distemper virus does adversely affect its immunogenic properties. This is avoided in this laboratory by periodically adapting fresh field strains and using a special technique in the preparation and preservation of the vaccine.

Furthermore, the process of freeze-drying the virus-vaccine under vacuum has prolonged its viability—a technique which was not available when the old killed-vaccine and virus or the serum and virus methods were used. This laboratory includes with the vaccine a suitable sterile syringe free from harmful chemicals liable to affect the viability of the virus at the time of inoculation.

Canine virus hepatitis. In my experience the incidence of this infection in puppies below the age 8 to 10 weeks is very rare, and for all practical purposes immunisation of dogs below this age would not stimulate an effective immunity.

In 1952 I commenced immunisation by the conjunctival instillation method and in all cases provoked a solid immunity. Dogs immunised 8 years ago can still withstand any challenge. A state of resistance to infection occurs within 24 hours after the use of this vaccine, which has enabled this method to be used for the protection of healthy incontacts during an outbreak. In a few cases a marked " eyelid reaction " has occurred which subsided normally without any resultant damage ever occurring. It must be borne in mind that the response to vaccination can vary even in a litter.

A killed vaccine, on its own, can never produce a level of immunity comparable to the use of the live virus. Over-attenuation of the virus will result in the same uncertainty of the period of immunity as Mr. Hanscomb remarked with the distemper immunisation.

Although under certain experimental conditions the reacting animal may spread infection to the incontact during the time of the reaction up to 11 to 15 days after vaccination. I have never found during 7 years' field trials that this method has caused directly or indirectly the spread of infection to dogs in neighbouring premises—houses, flats, cottages or kennels.

The strains of viruses we use in this laboratory were isolated from this country and are therefore of British origin.

I would welcome any independent enquiry by a competent veterinary virologist regarding the efficiency of our vaccines. I hope this will answer the many salient points brought forward by Mr. Hanscomb.

Yours faithfully,
W. MANSI.

Mansi Laboratories Ltd.,
Herons Way,
Wey Road,
Weybridge,
Surrey.

February 20th, 1960.

References

CABASSO, V. J. (1952). *Vet. Med.* **47**. 417.
MANSI, W. (1951). *Brit. Vet. J.* **108**. 14.

Sir,—I am sure many of us have followed the correspondence under the above heading with interest, and feel how right Messrs. Swift, Robertson, Macleod and Hoddinott are in bringing to notice this insidious danger of supplying drugs and injections to animal owners for use without professional diagnosis and advice.

I cannot see how the argument in Mr. Vaughan's letter (February 13th) justifies the practice at all.

The formulations and recommendations of certain concerns may be perfectly proper, but who makes the diagnoses? Without diagnosis the owner not only may waste his money giving drenches and what not, but wastes valuable time before proper treatment is instituted, thereby gravely lessening the chances of his animal making a quick and complete recovery.

The same principles and dangers exist regarding herd and flock prophylaxis. Surely a profit motive cannot justify such recklessness and injustice to animals?

UFAW have for some time been concerned at this injustice, and with the fact that, in its view, unnecessary suffering may be caused to animals as the result of treatment with "patent" medicine instead of medicine recommended as a result of professional attention.

UFAW will readily and actively support any move to control the sale and use of such products.

Yours faithfully,
T. HUGHES ELLIS,
President, Veterinary Section,
Universities Federation of Animal Welfare,
Compton House,
Chwilog,
Caernarvonshire.
February 23rd, 1960.

Sir,—We were pleased to see that our letter of January 23rd has received some support, and think that Mr. Hoddinott's letter of January 30th contains some points worthy of consideration.

He is correct when he lays part of the blame for the present state of affairs on our own profession. In the past many of us have ignored the supplying of vaccines, sera, anthelmintics, etc., and others, we believe, have not always been above overcharging. The result is that probably less than 20 per cent. of sheep biologicals now sold are distributed under veterinary supervision. Mr. Hoddinott suggests that legislation be introduced whereby no veterinary instrument or injectable drug may be purchased except from a veterinary surgeon. We think it may prove difficult to have such legislation introduced, but that an equally effective method of control would emerge if all the manufacturers would adopt the policy of selling only to the profession.

We understand from a pharmaceutical representative that discussions took place in 1950 or 1951 between the pharmaceutical industry and the veterinary profession regarding the sale and distribution of cattle, sheep, and pig biologicals. We were given to understand that recommendations were made to the effect that biologicals relating to cattle and pigs would be available only to the profession; those relating to sheep would be given a much wider distribution.

We are not in a position to probe the reasons for the 1950 findings which prompted those concerned to recommend a different procedure in the case of sheep biologicals, but, in our opinion, the state of the sheep industry has altered considerably since the time the negotiators met. Not only does sheep-farming play an ever-increasing rôle in the agricultural economy, but also we have found an alarming increase in the incidence of sheep diseases which necessitate the preventive use of biological products, *e.g.* enzootic abortion and pneumonia caused by *Pasteurella* spp.

We consider that the time has again arrived for a "Commission" to meet in order that the irregularities mentioned in our earlier letter be ironed out and the abuses stopped.

Yours faithfully,
G. MACLEOD,
W. W. ROBERTSON.

Dornoch,
Sutherland.
February 13th, 1960.

Treatment of Agalactia in Sows

Sir,—In connexion with a recent article by Noble, Marshall, and Oakley (*Vet. Rec.* **72**. 60), on the treatment of agalactia in sows using a combination of corticosteroid and antibiotic therapy, you may be interested in the results obtained by Ringarp, a veterinary surgeon in practice in south Sweden (Ringarp, 1960). Ringarp compared the piglet mortality rate at 3 weeks old in 3 groups of piglets:—

Group A. Controls (sows clinically healthy and with normal lactation).
Group B. Sows with agalactia toxaemia and treated by accepted methods (posterior pituitary hormone and anti-bacterial drugs).
Group C. Sows with agalactia toxaemia and treated as in Group B + prednisolone (100 mg. in severe cases and 50 to 60 mg. in mild cases).

He obtained the following results:—

Group	Number of piglets born	Three-week mortality rate as percentage of number of piglets born
A	762	17.3
B	683	30.6
C	640	23.0

The difference between mortality rates in groups B and C was statistically significant.

Yours faithfully,
HUGH BOYD.

"Blackpost Corner,"
Totnes,
Devon.
February 1st, 1960.

References

NOBLE, W. A., MARSHALL, A. A., & OAKLEY, G. (1960). *Vet. Rec.* **72**. 60.
RINGARP, N. (1960). *Acta Agriculturae Scandinavica.* Suppl. 7. Stockholm.

Whither Veterinary Surgeons?

Sir,—Dr. Abrams's article "Whither Veterinary Education?" in THE VETERINARY RECORD of February 13th, prompts me to ask the above question.

As an assistant veterinary surgeon the health of my colleagues in general practice seems, to me, to be in a perilous state at the moment; yet the monetary rewards to be gained from general practice are greater now than they ever have been, *viz.* the salary and "perks" offered to assistants in the columns of your journal.

At the present moment principals of practices are offering large salaries with free houses and unlimited use of cars during time off in return for services. By and large the assistant has to, and does, work long hours for this "pot of gold." At first all is well and then "practice fatigue" sets in. "Practice fatigue" is an occupational disease caused by a combination of any or all of the following:—

(1) Driving from 50 to 120 miles or more day after day with perhaps a break of one day per week during which the veterinary surgeon takes his family out in the car.

(2) Trying to keep up a high professional standard and at the same time complete 10 to 20 or more visits daily.

(3) Trying to keep to a time schedule, as demanded by the clients, and if this is upset by an emergency call, worrying, increasing car speed, and lowering diagnosis time to catch up.

(4) Being interrupted half-way through "Wagon Train" or some such relaxation treatment by a call to an animal which has been ill for the last 3 days.

(5) Other minor annoyances, *e.g.* curtailed lunch breaks, dirty bags for hand drying, etc.

With the onset of "practice fatigue" the Appointments Vacant columns of THE VETERINARY RECORD present more interesting reading than the rest of the journal.

Now the principal also is afflicted by "practice fatigue" but unfortunately he cannot have recourse to the Appointments Vacant treatment. Instead he deadens the pain with the palliative that from an increased output he can enjoy a *pro rata* increased income. The effect of this drug is very temporary. In addition to "practice fatigue" the principal usually falls victim to "administrative pyrexia." This is caused by:—

(1) Trying to keep up with Ministry instructions.

(2) Getting accounts out and in.

(3) Keeping drug stocks up to date.

(4) P.A.Y.E., N.I. stamps, etc.

The symptoms are generally restlessness in bed at night, hot and cold perspiration, loss of appetite, thinning of hair (if any) and a tendency to shout in the mornings.

Now what is going to be the outcome of this state of affairs? Besides the obvious physical and mental abnormalities that may arise, professional standards of clinical medicine and surgery are going to deteriorate. The young graduate will start off full of good intentions of enhancing the reputation of the profession, but with the onset of these chronic maladies the first steps on the road to hell are taken.

The answer? The immediate answer is to speed up the Veterinary Nursing Service upon which the R.C.V.S. and the B.V.A. have been working. In the Report of the Council of the R.C.V.S. for the year ended March 31st, 1959, we are told on page 31 that the Council has determined to press on with the scheme for animal or veterinary nurses, and has approached the Minister of Health direct.

I know that a lot of work may be going on behind the scenes about which the ordinary veterinary surgeon has no knowledge. But surely 12 months is rather a long time to wait without further information being made available. If the main problem is the term "nurse" may I suggest the designation "veterinary auxiliary"? At any rate more extensive lay help than is at present permitted is urgently required.

Veterinary auxiliaries could be trained in 3 to 6 months to carry out the dehorning of calves, the castrating of pigs and calves, cutting puppies' tails, and a whole series of minor jobs that are only tedious and time-consuming to the practising veterinary surgeon. The student auxiliary would do his training with a veterinary surgeon, and having completed his course his name would be put forward to the R.C.V.S. for examination by a veterinary surgeon appointed by them. This is on a parallel to the training of artificial inseminators licensed by the Ministry of Agriculture. A strict code of ethics and behaviour could be laid down to ensure that there would be no violation of the rights of veterinary surgeons or the welfare of the patients. "Veterinary auxiliaries" could be employed on a 5-day week, 9 a.m. to 5 p.m., and the burden that they would lift from the shoulders of veterinary surgeons would be immense.

I fully realise that the foregoing proposal would require an Act of Parliament, but if the R.C.V.S. were sufficiently interested that should prove no stumbling block.

I should be interested in the constructive views of other members of the profession.

Yours faithfully,
J. LAWSON.

106, New Road,
Bromsgrove,
Worcs.
February 15th, 1960.

Sensitivity to Antibiotics

Sir,—It would seem probable that more practices will be setting up bacteriological laboratories in order to give a better service to the poultry industry, and to be able to decide with more certainty which of the many antibiotics should be prescribed in individual cases of enteritis, mastitis, etc., in other species.

A large mass of data will be available on the antibiotic and chemical sensitivity of organisms recovered from "field" cases and it would be a great pity if no attempt were made to collect and analyse these findings.

Would it not be possible for some organisation—university, Animal Health Trust, Ministry of Agriculture, or commercial laboratory—to evolve some standard in technique and recording which could be used by all of us engaged in this side of practice?

Yours faithfully,
PETER J. DALTON.

Old Sarum House,
Stratford Road,
Salisbury.
February 19th, 1960.